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Plasma atrial natriuretic peptide vs. CVP for evaluation of preload to the heart during exercise in humans

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Introduction: In an upright posture, central venous pressure (CVP) becomes negative, in parallel with reduction in the central blood volume and, consequently, preload to the heart and cardiac output (CO) decreases (Harms et al. 2003). The influence of orthostatic reduction in CVP on preload to the heart is not established. During exercise leg muscles pump blood from the legs towards the heart (Beecher et al. 1936) as illustrated by enhanced plasma ANP, apparently with little influence on CVP (Vogelsang et al. 2006). We hypothesized that even though orthostasis reduces CVP also during exercise, the increased cardiac preload brings about an increase in CO.

Aim: This study evaluated the effect of the presumed different central venous pressure (CVP) response to running and rowing on preload to the heart. Preload was indicated by distension of the atrium as evaluated by plasma arterial natriuretic peptide (ANP) and related to cardiac output (CO).

Methods: Seven healthy subjects (four men; 26 ± 3 years; 181 ± 8 cm height; and 76 ± 11 kg, weight; mean \pm SD) performed, in random order, rowing on an ergometer (Concept II, Morrisville, VT, USA) and walking followed by running on a treadmill (Runrace, Technogym, Gambettola, Italy) at three intensities aiming at heart rate (HR) of 120, 140, and 160 beats per minute and lasting about 7 min each.

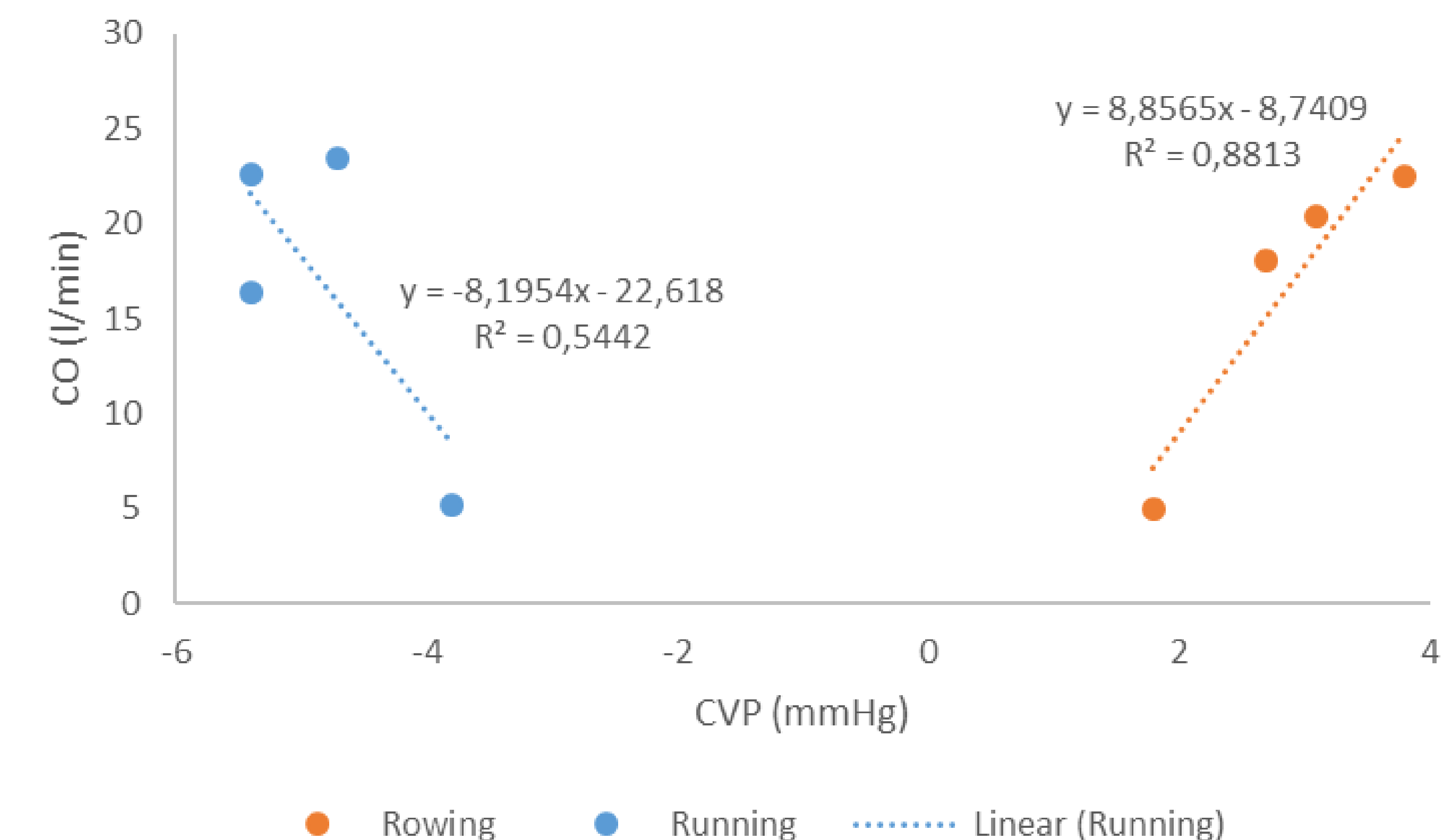
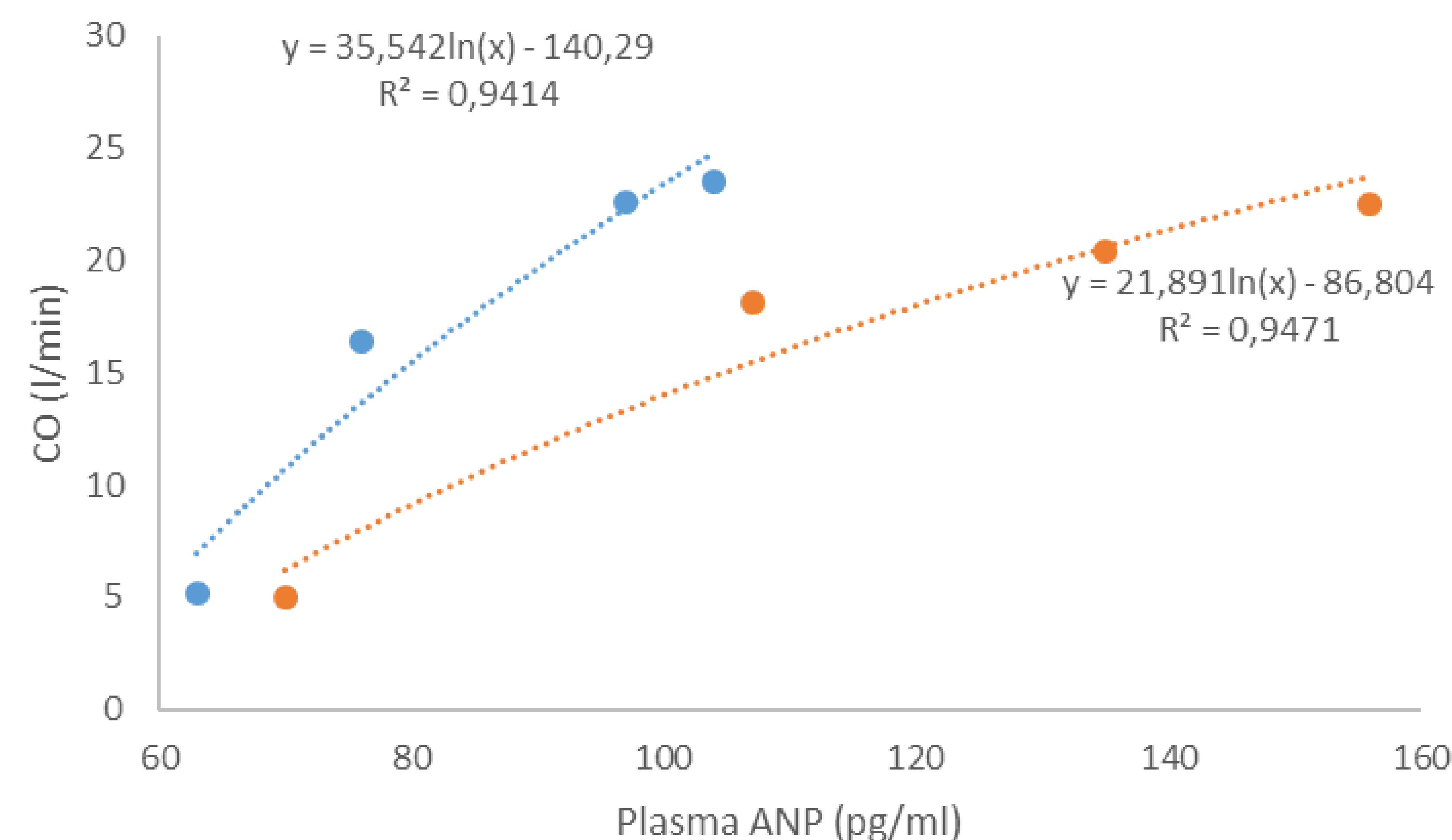
Cardiovascular variables

CVP was measured from a catheter advanced to the right atrium and mean arterial pressure (MAP) was determined from the radial artery. A modified Modelflow method estimated CO from the radial arterial pressure (Finapres Medical Systems, Amsterdam, the Netherlands). HR was recorded from a three-lead electrocardiogram (Medicotest Q-10-A, Copenhagen, Denmark).

Blood samples from the radial artery and the central venous catheter were analyzed for pH, oxygen saturation (SO_2) and tension (PO_2) (ABL, Radiometer, Copenhagen, Denmark). Also, arterial samples (in EDTA tubes) were analyzed for ANP by radioimmunoassay (Schütten et al. 1987).



Results: While sitting CO was 6.2 ± 1.6 l/min, plasma ANP 70 ± 10 pg/ml, and CVP 1.8 ± 1.1 mmHg (mean \pm SD) and decreased to 5.9 ± 1.0 l/min, 63 ± 10 pg/ml, and -3.8 ± 1.2 mmHg when standing ($P < 0.05$). Ergometer rowing elicited an increase in CO to 22.5 ± 5.5 l/min as plasma ANP increased to 156 ± 11 pg/ml and CVP to 3.8 ± 0.9 mmHg ($P < 0.05$). Similarly, CO increased to 23.5 ± 6.0 l/min during running with albeit smaller ($P < 0.05$) increase in plasma ANP, but with little change in CVP (around -0.9 ± 0.4 mmHg)



Figures. Logarithmic and linear regressions between cardiac output (CO), plasma atrial natriuretic peptide (ANP, left) and central venous pressure (CVP, above) at rest and during three exercise intensities for running and rowing ($n = 7$, $P < 0.05$).

Conclusion: During exercise the increase in CO is curvilinearly related to plasma ANP, as an indication of preload to the heart, but it is unrelated to CVP. The results indicate that in the upright posture CVP reflects the gravitational influence on central venous blood and not preload to the heart.

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